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ACC/AHA TASK FORCE REPORT

Guidelines for Clinical Intracardiac Electrophysiological and Catheter Ablation Procedures

A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Clinical Intracardiac Electrophysiologic and Catheter Ablation Procedures), Developed in Collaboration With the North American Society of Pacing and Electrophysiology

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Preamble

It is becoming apparent that despite a strong national commitment to excellence in health care, resources and personnel are finite. It is therefore appropriate that the medical profession examine the impact of developing technology and new therapeutic modalities on the practice of cardiology. Such analysis, carefully conducted, could potentially impact the cost of medical care without diminishing the effectiveness of that care.

To this end, the American College of Cardiology and the American Heart Association in 1980 established a Task Force on the Assessment of Diagnostic and Therapeutic Cardiovascular Procedures (now the ACC/AHA Task Force on Practice Guidelines) with the following charge:

> The task force of the American College of Cardiology and the American Heart Association shall develop guidelines relative to the role of new therapeutic approaches and specific noninvasive and invasive procedures in the diagnosis and management of cardiovascular disease.
>
> The task force shall address, when appropriate, the contribution, uniqueness, sensitivity, specificity, indications, contraindications and cost-effectiveness of such diagnostic procedures and therapeutic modalities.
>
> The task force shall emphasize the role and value of the developed guidelines as an educational resource.
>
> The task force shall include a chair and six members, three representatives from the American Heart Association

"Guidelines for Clinical Intracardiac Electrophysiological and Catheter Ablation Procedures" was approved by the American Heart Association SAC/Steering Committee in May 1995 and the American College of Cardiology Board of Trustees in March 1995 and was endorsed by the North American Society of Pacing and Electrophysiology Board of Directors in May 1995.

This statement is being published simultaneously in *Circulation*, the *Journal of the American College of Cardiology*, and the *Journal of Cardiovascular Electrophysiology*.

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and three representatives from the American College of Cardiology. The task force may select ad hoc members as needed upon the approval of the presidents of both organizations. Recommendations of the task force are forwarded to the president of each organization.

The members of the ACC/AHA Task Force on Practice Guidelines are Melvin D. Cheitlin, MD, FACC, Arthur Garson, Jr., MD, MPH, FACC, Raymond J. Gibbons, MD, FACC, Richard P. Lewis, MD, FACC, Robert A. O'Rourke, MD, FACC, Thomas J. Ryan, MD, FACC, Robert C. Schlant, MD, FACC, and James L. Ritchie, MD, FACC, chair.

The Committee on Clinical Intracardiac Electrophysiologic Studies was chaired by Douglas P. Zipes, MD, FACC, and included the following members: John P. DiMarco, MD, PhD, FACC, Paul C. Gillette, MD, FACC, Warren M. Jackman, MD, FACC, Robert J. Myerburg, MD, FACC, and Shahbudin H. Rahimtoola, MD, FACC. The committee would like to thank John D. Fisher, MD, FACC, for his contributions to section XV, "Indications for Catheter Ablation Procedures."

This document was reviewed by the officers and other responsible individuals of the American College of Cardiology and the American Heart Association and received final approval in May 1995. This document also was reviewed and endorsed by the officers of the North American Society for Pacing and Electrophysiology. It is being published simultaneously in *Circulation*, the *Journal of the American College of Cardiology*, and the *Journal of Cardiovascular Electrophysiology*.

James L. Ritchie, MD, FACC
Chair, ACC/AHA Task Force on Practice Guidelines

Executive Summary

ACC/AHA guidelines for clinical intracardiac electrophysiologic studies were first published in 1989. Since then significant changes have occurred, and this report is an update of the initial publication. It is divided into 12 sections on the value of electrophysiological studies for diagnosis and assessment of therapy for bradyarrhythmias and tachyarrhythmias, with additional sections on the use of electrophysiological studies in patients with implantable electrical devices, indications for catheter ablation, and special considerations for pediatric patients. Indications for procedures are listed as Class I, Class II, and Class III.

Bradyarrhythmias

The use of electrophysiological studies in patients with known or suspected bradyarrhythmias has been divided into sections on evaluation of patients with sinus node dysfunction, atrioventricular (AV) block, and intraventricular conduction delay. For most of these patients, electrophysiological studies are viewed as supplements to the analysis of standard electrocardiographic (ECG) recordings, which, in most patients, are adequate for diagnosis and clinical decisions. Electrophysiological studies provide useful information when ECG data are nondiagnostic or unobtainable. Possible roles for electrophysiological procedures in patients before pacemaker implantation are also discussed in the section on implantable devices.

Tachyarrhythmias

The role of electrophysiological studies in patients with known or suspected tachyarrhythmias is covered in sections on diagnostic and prognostic use in patients with narrow and wide complex tachycardias, long QT intervals, Wolff-Parkinson-White syndrome, and syncope of unknown cause. There is also a section on use of electrophysiological studies for evaluation of antiarrhythmic drug therapy. In general, electrophysiological data are considered the gold standard for diagnosis of most tachyarrhythmias, and their use is recommended whenever ECG diagnosis of an arrhythmia of clinical significance is uncertain. The value and, importantly, the limitations of electrophysiological studies in assessing prognosis and therapy are also discussed. Electrophysiological studies are considered intrinsic in the prescription and evaluation of implantable cardioverter-defibrillators for therapy of ventricular tachyarrhythmias. The advent of radiofrequency catheter ablation has expanded the use of electrophysiological procedures to include therapy. These guidelines now have sections on the value of catheter ablation in patients with paroxysmal supraventricular tachycardia, preexcitation syndromes, ventricular tachycardia, and atrial tachyarrhythmias such as atrial tachycardia, atrial flutter, and atrial fibrillation. The guidelines state that catheter ablation is one of the primary treatment options for most forms of paroxysmal supraventricular tachycardias and preexcitation syndromes, monomorphic ventricular tachycardia and structurally normal hearts. Use of catheter ablation is justified by the high success and low complication rates described in the literature. For other tachyarrhythmias, the guidelines recommend only selective use of catheter ablation because of limited data about efficacy during long-term follow-up. However, the rapid expansion of knowledge in these areas is recognized. Catheter procedures to ablate or modify atrioventricular conduction are recognized as effective in controlling ventricular rates in patients with atrial tachyarrhythmias. In the future, ablation may be used to eliminate atrial fibrillation.

I. Introduction

During the past 25 years, cardiac electrophysiological studies have become widely used clinical tools, often indispensable in evaluating patients with specific cardiac arrhythmias. Because such studies carry a relatively small but finite risk of major as well as minor complications, routinely involve the purposeful induction of serious arrhythmias, and consume healthcare resources, it is important that their clinical usefulness for diagnosis and therapy of cardiac arrhythmias be carefully considered. These guidelines present current opinion regarding clinical application of invasive cardiac electrophysiological studies and update earlier publications on this subject.[1-3] The American College of Cardiology, the American Heart Association, and the

North American Society of Pacing and Electrophysiology recognize that the ultimate judgment regarding the appropriateness of a specific procedure is the responsibility of the physician caring for the patient. Therefore, these guidelines should not be considered all inclusive or exclusive of other methods that may be available for the care of the individual patient. Moreover, these guidelines address in only a limited way the cost-benefit and risk-benefit outcomes of these procedures. Finally, it is important to emphasize that this document, like the one before it,[3] will require periodic updating as the indications for electrophysiological studies continue to evolve as a result of increasing knowledge and technological advances.

It is assumed, for the purposes of this document, that the electrophysiological studies are performed by appropriately trained and qualified personnel in adequately equipped laboratories, and that complete electrophysiological studies, indicated by the patient's clinical state and specific arrhythmia, are performed. In general, such studies include intravenous or intra-arterial placement, or both, of one or more electrode catheters at one or more sites in the atria, ventricles, or coronary sinus (occasionally in the pulmonary artery or aorta) or esophagus to record or stimulate at various rates and cadences. Such studies are performed to evaluate electrophysiological properties such as automaticity, conduction, and refractoriness; initiate and terminate tachycardias; map activation sequences; evaluate patients for various forms of therapy; and judge response to therapy. Studies are modified according to the patient, the problem being investigated, and the site of the study, ie, bedside, operating room, or electrophysiology laboratory. Increasingly, in the course of these studies, therapeutic interventions such as catheter ablation procedures are being performed.

The indications for electrophysiological studies have been divided into three classes:

Class I

Conditions for which there is general agreement that the electrophysiological study provides information that is useful and important for patient treatment. Experts agree that patients with these conditions are likely to benefit from electrophysiological studies.

Class II

Conditions for which electrophysiological studies are frequently performed, but there is less certainty about the usefulness of the information that is obtained. Experts are divided in their opinion as to whether patients with these conditions are likely to benefit from electrophysiological study.

Class III

Conditions for which there is general agreement that electrophysiological studies do not provide useful information. Experts agree that electrophysiological studies are not warranted in patients with these conditions. This classification is assigned to patients with a variety of arrhythmias and clinical syndromes resulting from cardiac electrical abnormalities. Because use of electrophysiological studies in children on occasion differs from that in adults, it is discussed in another section.

II. Role of Electrophysiological Study in Evaluating Sinus Node Function

Electrocardiographic manifestations of sinus node dysfunction may include resting sinus bradycardia, inappropriate chronotropic responses to exercise or stress, sinoatrial exit block, or sinus arrest. Common manifestations include syncope, near syncope, transient lightheadedness, or severe fatigue. These arrhythmias are commonly caused by one or more of the following: intrinsic disease within the sinus node (eg, idiopathic degeneration, fibrosis, ischemia, or surgical trauma), abnormalities in autonomic nervous system function, and drug effects. Clinical evaluation of suspected sinus node dysfunction is often difficult because symptomatic abnormalities may be transient and healthy, asymptomatic persons can exhibit considerable variability in sinus rates.

Several invasive tests of sinus node function have been proposed. Sinus node recovery time (SNRT) is used to evaluate the effects of overdrive suppression on sinus node automaticity.[4] This is often corrected for the underlying baseline sinus cycle length (SCL) and expressed as corrected sinus node recovery time (CSNRT=SNRT−SCL). Sinoatrial conduction can be estimated indirectly either by introducing atrial extrastimuli during sinus rhythm[5,6] or by atrial pacing.[7] Catheter recordings of sinus node electrograms have been reported, and such direct measurements of conduction times seem to correlate well with the indirect measures described above.[8,9]

Sinus node recovery times and sinoatrial conduction times are frequently abnormal in symptomatic patients with intrinsic sinus node disease but will usually be normal in patients with sinus bradyarrhythmias caused by intermittent factors such as autonomic nervous system influences.[10,11] A full evaluation of sinus node function frequently requires continuous or intermittent ambulatory ECG recordings, exercise testing to assess chronotropic competence, tilt-table testing, or autonomic manipulations as well as invasive electrophysiological data.[12] In addition, patients with sinus node dysfunction may also be subject to other arrhythmias that may be investigated during an electrophysiological study.

Recommendations for Electrophysiological Studies

Class I

Symptomatic patients in whom sinus node dysfunction is suspected as the cause of symptoms but a causal relation between an arrhythmia and the symptoms has not been established after appropriate evaluation

Class II

(1) Patients with documented sinus node dysfunction in whom evaluation of atrioventricular or ventriculoatrial (VA) conduction or susceptibility to arrhythmias may aid in selection of the most appropriate pacing modality

(2) Patients with electrocardiographically documented sinus bradyarrhythmias to determine if abnormalities are due to intrinsic disease, autonomic nervous system dysfunction, or the effects of drugs so as to help select therapeutic options

(3) Symptomatic patients with known sinus bradyar-

rhythmias to evaluate potential for other arrhythmias as the cause of symptoms

Class III

(1) Symptomatic patients in whom an association between symptoms and a documented bradyarrhythmia has been established and choice of therapy would not be affected by results of an electrophysiological study

(2) Asymptomatic patients with sinus bradyarrhythmias or sinus pauses observed only during sleep, including sleep apnea

III. Role of Electrophysiological Study in Patients With Acquired Atrioventricular Block

The electrocardiographic classification of AV block includes these categories:

(1) First-degree AV block— prolongation of the PR interval beyond 0.20 second.

(2) Second-degree AV block—intermittent failure to conduct a single P wave. Two types have been described. In type I second-degree AV block (Wenckebach block) there is progressive prolongation of the PR interval before the blocked P wave; in type II AV block PR intervals are constant before the blocked P wave.

(3) AV block with a 2:1 conduction ratio—not classified as either type I or type II but as 2:1 AV block.

(4) Advanced or high-grade AV block—this category is recommended by some experts to define a condition in which multiple consecutive P waves are blocked but complete AV block is not present.

(5) Complete AV block—failure of all P waves to conduct to the ventricle, resulting in complete dissociation between P waves and QRS complexes.

His bundle recordings allow delineation of three anatomic sites of AV block[13]: (1) proximal (above the His bundle), representing delay or block in the AV node; (2) intra-Hisian, representing delay or block within the His bundle; and (3) infra-Hisian or distal to the His bundle, representing block or delay distal to the His bundle recording site either in the distal His bundle or the bundle branches.

There are certain correlations between ECG patterns and the site of block.[14] In type I second-degree AV block with narrow QRS complexes, the block is usually at the level of the AV node; less frequently it may be within the His bundle. In type I second-degree AV block with wide QRS complexes (bundle branch block), the block may be in the AV node or within or below the His bundle. Type II second-degree AV block is usually within or below the His bundle and is most often seen with bundle branch block. Rarely, type II AV block can be in the AV node. In complete AV block with an escape rhythm of narrow QRS complexes, the site of block may be in the AV node or within the His bundle. In complete AV block with an escape rhythm of wide QRS complexes, the site of block may be in the AV node or within or below the His bundle; usually it is below the His bundle. Clinical information about age, gender, underlying heart disease, and the use of cardiac medications may also be helpful in predicting the site of AV block.

The prognosis of patients with AV block depends on the site of block. Chronic first-degree AV block, particularly AV nodal block, usually has a good prognosis. The abnormality is frequently drug related and reversible. The clinical course of patients with second-degree AV nodal block is usually benign, and prognosis depends on the presence and severity of underlying heart disease.[15] The prognosis of patients with second-degree AV block within the His bundle is uncertain. Such patients frequently manifest congestive heart failure and syncope. Untreated chronic second-degree AV block below the His bundle has a poor prognosis; patients frequently proceed to higher degrees of block and become symptomatic with syncope.[16] Patients with untreated, acquired complete AV block are often symptomatic regardless of the site of the block.[17]

Recommendations for Electrophysiological Studies

Class I

(1) Symptomatic patients in whom His-Purkinje block, suspected as a cause of symptoms, has not been established

(2) Patients with second- or third-degree AV block treated with a pacemaker who remain symptomatic and in whom another arrhythmia is suspected as a cause of symptoms

Class II

(1) Patients with second- or third-degree AV block in whom knowledge of the site of block or its mechanism or response to pharmacological or other temporary intervention may help direct therapy or assess prognosis

(2) Patients with premature, concealed junctional depolarizations suspected as a cause of second- or third-degree AV block pattern (ie, pseudo AV block).

Class III

(1) Symptomatic patients in whom the symptoms and presence of AV block are correlated by ECG findings

(2) Asymptomatic patients with transient AV block associated with sinus slowing (eg, nocturnal type I second-degree AV block).

IV. Role of Electrophysiological Study in Patients With Chronic Intraventricular Conduction Delay

As judged from the electrocardiogram (ECG), the intraventricular conduction system appears to be trifascicular, consisting of the two fascicles of the left bundle (anterior and posterior) and the right bundle branch. The anatomic basis for the trifascicular conduction system in humans is less clearly defined. The HV interval in patients with bifascicular block is a measure of the conduction time through the remaining functioning fascicle. Most data on the importance of the HV interval in predicting subsequent development of AV block have been derived from patients with bifascicular block. Patients with bifascicular block and a prolonged HV interval (>55 milliseconds) appear to have a slightly increased risk of developing complete trifascicular block.[18,19] Although the prevalence of prolonged HV interval is high, the incidence of complete trifascicular block is low (2% to 3% annually but greater if the HV interval exceeds 100 milliseconds), and the rate of progression in the absence of an acute intervening event (drugs, electrolyte abnormalities, or ischemia) is low.[18-20] Thus, the HV

interval has a high sensitivity (82%) but a low specificity (63%) for predicting development of complete trifascicular block.[18]

Rapid atrial pacing has been used to improve the specificity of electrophysiological testing in patients with bifascicular block. An abnormal response consists of the development of block distal to the His bundle with rapid atrial pacing during 1:1 AV nodal conduction. Functional block distal to the His bundle due to abrupt shortening of the coupling interval (such as during the long-short cycles of Wenckebach periods or at the onset of pacing) is not considered a positive response. The sensitivity of distal His block induced by atrial pacing is relatively low, but its positive predictive value for development of complete AV block is high.[21]

Sudden death in patients with bifascicular block may not be caused by the development of complete trifascicular block but rather the presence of ventricular tachyarrhythmias.[22] The latter may play a significant role in patients with advanced heart disease and bifascicular block. For this reason, electrophysiological evaluation of patients with intraventricular conduction defects and unexplained symptoms should also include study of the AV conduction system and evaluation of sinus node function and programmed atrial and ventricular stimulation to assess the propensity for development of both bradyarrhythmias and tachyarrhythmias in an attempt to induce tachyarrhythmias.

Recommendations for Electrophysiological Studies

Class I

Symptomatic patients in whom the cause of symptoms is not known

Class II

Asymptomatic patients with bundle branch block in whom pharmacological therapy that could increase conduction delay or produce heart block is contemplated

Class III

(1) Asymptomatic patients with intraventricular conduction delay

(2) Symptomatic patients whose symptoms can be correlated with or excluded by ECG events

V. Role of Electrophysiological Study in Diagnosis of Patients With Narrow QRS Complex Tachycardias

A narrow QRS tachycardia (QRS complex <120 milliseconds) can be caused by impulse formation in the sinus node (sinus tachycardia), a reentry circuit in the sinus node or the sinus node and a part of the contiguous atrium (sinus node reentry), in the atrium (atrial tachycardia, atrial flutter, and atrial fibrillation), in the AV node–His bundle axis (AV junctional tachycardia), reentry involving the AV node and its approaches (AV nodal reentrant tachycardia), and reentry by the AV node–His pathway for anterograde AV conduction and an accessory AV pathway for retrograde conduction (orthodromic AV reentrant tachycardia [AVRT]). Less commonly, impulse formation in the intraventricular specialized conduction system can lead to a ventricular tachycardia (VT) with a QRS complex less than 120 milliseconds in duration (fascicular tachycardia).[23]

Frequently, careful examination of the 12-lead ECG, especially when a recording during carotid sinus massage or other vagal maneuvers is also available, facilitates making the correct diagnosis. Proper identification of the site of origin of atrial activity, its rate, and its relation to the ventricular rhythm is essential.

During a typical atrial tachycardia, atrial activity precedes each QRS complex. While the PR interval may vary according to the conduction capabilities of the AV node and the rate of the atrial tachycardia, usually the P wave is located in the second half of the tachycardia cycle, creating a PR interval shorter than the RP interval. Importantly, the atrial tachycardia can continue despite the development of AV block because activation of the ventricles is not an obligatory part of the tachycardia circuit. Four other narrow QRS tachycardias also create an RP interval that usually exceeds the PR interval, including sinus node reentry, inappropriate sinus tachycardia, atypical AV nodal reentry, and the permanent form of AV junctional reciprocating tachycardia (PJRT). AV block with continuation of tachycardia can also occur in sinus node reentry, sinus tachycardia, and atypical AV nodal reentry. Anterograde conduction over the fast conducting AV nodal pathway and retrograde conduction over the slowly conducting AV nodal pathway provide the reentrant circuit for atypical AV nodal reentry,* while anterograde conduction over the AV node and retrograde conduction over a slowly conducting accessory pathway constitute the circuit for PJRT. Some patients in whom the retrograde P wave is located midway in the cardiac cycle have AV nodal reentry with two slowly conducting pathways, one anterogradely and the other retrogradely, so called "slow-slow" AV node reentry.

In typical AV nodal reentrant tachycardia, the atria and ventricles are activated simultaneously due to anterograde conduction over the slowly conducting pathway and retrograde conduction over the fast conducting pathway. The retrograde P wave is obscured by the QRS complex or inscribed in the terminal portion of the QRS complex.[24,25] AV block with continuation of the tachycardia can occur. During orthodromic AVRT, the tachycardia circuit is formed by anterograde conduction over the AV node and retrograde conduction over an accessory AV pathway; retrograde atrial activation follows the QRS complex, and the P wave is located in the ST segment. The tachycardia cannot continue in the presence of AV block. In both AV nodal reentry and orthodromic AVRT, the retrograde P wave occurs in the first half of the tachycardia cycle so that the RP interval is shorter than the PR interval.

AV dissociation can be present during a narrow QRS tachycardia. When this happens, the tachycardia most commonly originates in the AV node–His bundle or fascicles of the ventricles.

*We have chosen to use the term *AV nodal reentry* rather than *AV junctional reentry* to avoid confusion with other tachycardias, despite the fact that the atrial approaches to the AV node are probably involved in this tachycardia.

Recommendations for Electrophysiological Studies

Class I

(1) Patients with frequent or poorly tolerated episodes of tachycardia that do not adequately respond to drug therapy and for whom information about site of origin, mechanism, and electrophysiological properties of the pathways of the tachycardia is essential for choosing appropriate therapy (drugs, catheter ablation, pacing, or surgery)

(2) Patients who prefer ablative therapy to pharmacological treatment (see section XV)

Class II

Patients with frequent episodes of tachycardia requiring drug treatment for whom there is concern about proarrhythmia or the effects of the antiarrhythmic drug on the sinus node or AV conduction

Class III

Patients with tachycardias easily controlled by vagal maneuvers and/or well-tolerated drug therapy who are not candidates for nonpharmacological therapy

VI. Role of Electrophysiological Study in Diagnosis of Patients With Wide QRS Complex Tachycardias

Wide QRS complex tachycardias (≥120 milliseconds in adults) can be caused by supraventricular arrhythmias with fixed or rate-related aberrant intraventricular conduction, supraventricular arrhythmias with anterograde preexcitation, and ventricular arrhythmias. Although numerous authors have proposed ECG criteria to differentiate supraventricular tachycardia with aberration from ventricular tachycardia,[26-29] this distinction may remain difficult even if complete ECG tracings are available for analysis. Arrhythmias with anterograde preexcitation may be particularly difficult to differentiate from VT based on QRS morphology criteria during tachycardia alone. Some arrhythmias, such as bundle branch reentry or atriofascicular tract tachycardias, have QRS patterns that are not different from those in the more commonly encountered forms of supraventricular tachycardia with aberration. Finally, some fraction of VTs may have QRS durations of ≤120 milliseconds with an abnormal QRS morphology,[30] and in pediatric patients VT QRS duration can be less than 120 milliseconds. Electrophysiological studies permit accurate diagnosis of virtually all wide complex tachycardias, and the sequence of and relation between atrial and ventricular activation can be determined. Electrograms from structures not represented on the surface ECG (eg, the His bundle or accessory pathways) can be recorded and responses to various pacing maneuvers can be analyzed. Because knowledge of the mechanism of arrhythmia is often critical for selecting appropriate therapy, electrophysiological studies for diagnosis are frequently of significant clinical value in patients with wide complex tachycardias.

Recommendations for Electrophysiological Studies

Class I

Patients with wide QRS complex tachycardia in whom correct diagnosis is unclear after analysis of available ECG tracings and for whom knowledge of the correct diagnosis is necessary for patient care

Class II

None

Class III

Patients with VT or supraventricular tachycardia with aberrant conduction or preexcitation syndromes diagnosed with certainty by ECG criteria and for whom invasive electrophysiological data would not influence therapy. However, data obtained at baseline electrophysiological study in these patients might be appropriate as a guide for subsequent therapy (see sections on therapy).

VII. Role of Electrophysiological Study in Patients With Prolonged QT Intervals

Prolongation of the QT interval and its association with potentially life-threatening ventricular tachyarrhythmias may occur chronically or intermittently as part of the congenital prolonged QT interval syndrome or may be acquired secondary to metabolic, toxic, or pathophysiological factors.[31] The autonomic nervous system and catecholamines can influence expression and manifestations of both congenital and acquired long QT interval syndromes.[32]

The role of electrophysiological studies in diagnosis and evaluation of, or guiding therapy for, either congenital or acquired QT prolongation is limited.[33] Electrophysiological studies in patients with congenital long QT syndrome infrequently result in initiation of ventricular arrhythmias[32,34,35] and are of limited or no predictive value.[34] Catecholamine infusion during electrophysiological study or continuous ECG monitoring has been proposed as a method of unmasking clinically subtle forms of prolonged QT interval syndromes in symptomatic patients,[32] but the positive and negative predictive accuracies of these methods have not been fully defined. Electrophysiological studies have been used for diagnostic purposes in patients who have unexplained syncope or symptomatic arrhythmias with acquired prolongation of the QT interval while receiving drugs with the potential for inducing torsade de pointes. However, the prognostic significance of drug-induced changes in the pattern of inducibility of ventricular arrhythmias remains uncertain.[36] Monophasic action potential recordings may provide measures of action potential durations and identification of afterdepolarizations[32,37,38] but at present are limited by the technical difficulty of achieving stable and reproducible recordings from defined sites.

Recommendations for Electrophysiological Studies

Class I

None

Class II

(1) Identification of a proarrhythmic effect of a drug in patients experiencing sustained VT or cardiac arrest while receiving the drug

(2) Patients who have equivocal abnormalities of QT interval duration or TU wave configuration, with syncope

or symptomatic arrhythmias, in whom catecholamine effects may unmask a distinct QT abnormality

Class III

(1) Patients with clinically manifest congenital QT prolongation, with or without symptomatic arrhythmias

(2) Patients with acquired prolonged QT syndrome with symptoms closely related to an identifiable cause or mechanism

VIII. Role of Electrophysiological Study in Patients With Wolff-Parkinson-White Syndrome

In Wolff-Parkinson-White (WPW) syndrome, patients are symptomatic from tachyarrhythmias caused by an anomalous AV connection (accessory pathway) that causes ventricular preexcitation and participates in supraventricular arrhythmias. The prevalence of ventricular preexcitation is thought to be 0.1% to 0.3% in the general population. Estimates of arrhythmia incidence in patients with preexcitation vary widely, ranging from 12% to 80% in several surveys.[39-41]

AVRT is the most common arrhythmia in patients with WPW syndrome. This tachycardia is further classified as either orthodromic AVRT, which occurs in 70% of symptomatic patients,[42] or antidromic AVRT, which occurs in only 4% to 5% of patients.[43-45] During orthodromic AVRT, the reentrant impulse propagates from atrium to ventricle by way of the normal conduction system (AV node and His-Purkinje system) and then propagates retrogradely to the atrium over the accessory pathway. During antidromic AVRT the reentrant impulse propagates from atrium to ventricle by conduction over the accessory pathway and retrogradely to the atrium by either the normal conduction system (His-Purkinje system and AV node) or another accessory pathway.

The prevalence of atrial fibrillation, the second most common arrhythmia in patients with WPW syndrome, ranges from 10% to 38%.[42,46,47] Atrial fibrillation can be complicated by a very rapid ventricular response (via conduction over the accessory pathway), which can lead to ventricular fibrillation. Patients with WPW syndrome and a history of ventricular fibrillation are more likely to have a history of both AVRT and atrial fibrillation, multiple accessory pathways, and rapid conduction over the accessory pathway during atrial fibrillation (shortest preexcited RR interval of <250 milliseconds during electrophysiological study).[48] The incidence of sudden cardiac death in patients with WPW syndrome has been estimated at 0.15% per patient year and is probably even lower in asymptomatic patients with ventricular preexcitation.[49]

Electrophysiological study can be used in patients with WPW syndrome to determine the mechanism of the clinical arrhythmia, electrophysiological properties (such as conduction capability and refractory periods) of the accessory pathway and the normal conduction system, number and location of accessory pathways (which is necessary for catheter ablation), and response to pharmacological or ablation therapy.

Recommendations for Electrophysiological Studies

Class I

(1) Patients being evaluated for catheter ablation or surgical ablation of an accessory pathway

(2) Patients with ventricular preexcitation who have survived cardiac arrest or who have unexplained syncope

(3) Symptomatic patients in whom determination of the mechanism of arrhythmia or knowledge of the electrophysiological properties of the accessory pathway and normal conduction system would help in determining appropriate therapy

Class II

(1) Asymptomatic patients with a family history of sudden cardiac death or with ventricular preexcitation but no spontaneous arrhythmia who engage in high-risk occupations or activities and in whom knowledge of the electrophysiological properties of the accessory pathway or inducible tachycardia may help determine recommendations for further activities or therapy

(2) Patients with ventricular preexcitation who are undergoing cardiac surgery for other reasons

Class III

Asymptomatic patients with ventricular preexcitation, except those in Class II above.

IX. Role of Electrophysiological Study in Patients With Premature Ventricular Complexes, Couplets, and Nonsustained Ventricular Tachycardia

Frequent or repetitive ventricular ectopy and nonsustained ventricular tachycardia (three or more consecutive ventricular complexes that last less than 30 seconds and do not produce loss of consciousness) can occur in patients with structurally normal and abnormal hearts. In treating these patients the clinician must consider both symptoms caused by the ventricular ectopy itself and the prognostic significance of these arrhythmias.

Patients with frequent or repetitive ventricular ectopy may experience symptoms such as palpitations, fatigue, and near-syncope in association with these arrhythmias. If the symptoms are mild or infrequent, it may be possible to avoid therapy. In patients in whom symptoms are poorly tolerated, electrophysiological study may be used to locate the site(s) of origin of the arrhythmia. If identified, the site can be treated with catheter ablation; documentation of recurrent ventricular complexes with a single discrete ECG morphology as the cause of symptoms is usually required. This approach has been highly successful in patients with VTs and structurally normal hearts.[50,51]

Frequent or complex ventricular ectopy has also been associated with an adverse prognosis in some clinical situations.[52] The coexistence of other prognostic variables, including the presence and type of structural heart disease, severity of ventricular dysfunction, abnormalities on a signal-averaged ECG, and loss of normal heart rate variability may also be used to assess prognosis.[53-57] In patients with coronary artery disease, clinical trials have tested the hypothesis that pharmacological therapy directed at suppression of ventricular ectopy reduces incidence of sudden

death; no benefit from such therapy has been shown.[58,59] Indeed, in the Cardiac Arrhythmia Suppression Trial, suppression of ventricular ectopy using flecainide, encainide, or moricizine was associated with increased mortality.[60]

A number of studies have assessed the value of programmed ventricular stimulation for estimating risk of future arrhythmic and total mortality.[57,61-70] These studies varied widely in methods of patient selection, stimulation protocols used, and definitions of relevant end points for stimulation. Data from patient groups with disorders other than prior myocardial infarction (MI) are too limited to provide clinical guidance. Among patients with prior MI, ability to induce a sustained monomorphic VT with programmed stimulation is associated with a greater than twofold risk of arrhythmia-related death during follow-up.[71] Controlled clinical trials are now under way to test the hypothesis that induction of VT and subsequent suppression of induction will effectively identify patients at highest risk and modify that risk with therapy.[72]

Recommendations for Electrophysiological Studies

Class I

None

Class II

(1) Patients with other risk factors for future arrhythmic events, such as a low ejection fraction, positive signal-averaged ECG, and nonsustained VT on ambulatory ECG recordings in whom electrophysiological studies will be used for further risk assessment and for guiding therapy in patients with inducible VT

(2) Patients with highly symptomatic, uniform morphology premature ventricular complexes, couplets, and nonsustained VT who are considered potential candidates for catheter ablation

Class III

Asymptomatic or mildly symptomatic patients with premature ventricular complexes, couplets, and nonsustained VT without other risk factors for sustained arrhythmias

X. Role of Electrophysiological Study in Patients With Unexplained Syncope

Syncope, near-syncope, and transient lightheadedness are common medical problems. Before the use of head-up tilt testing to identify a neurocardiogenic cause of syncope, the results of a large prospective study revealed that approximately 50% of patients had a definable cause of syncope: half cardiovascular and half noncardiovascular. The causes in the remaining 50% remained obscure.[73] Based on subsequent observations using head-up tilt testing, it is now recognized that in many in the latter group, plus some in the noncardiovascular group, abnormal neurocardiac reflexes was the underlying mechanism.[74] In the absence of manifest cardiac arrhythmias or structural cardiac disease, neurocardiogenic syncope now appears to be a common cause of unexplained syncope.[75,76] However, electrophysiological studies continue to be used to detect arrhythmias as a possible cause of syncope in persons at risk for or with manifestations of cardiac disease.[77-85] Syncope in the presence of cardiovascular disease heralds a much higher mortality risk than syncope without structural heart disease. A 12-lead ECG is not likely to provide specific etiologic or prognostic information that is useful for guiding therapy, but it may yield insight into the nature of underlying structural heart disease, the relevance of which may be defined during further diagnostic studies.

In the presence of structural heart disease, arrhythmias are a primary consideration among causes of syncope. Long-term ambulatory ECG recordings, head-up tilt testing, or exercise testing, alone or in combination, can be useful, but in patients with suspected ventricular arrhythmias, these tests should not necessarily precede or supplant electrophysiological studies. Syncope or near-syncope associated with ventricular tachyarrhythmias, rapid supraventricular tachycardias, or transient bradyarrhythmic events are sporadic, and thus continuous ambulatory recordings are often unrewarding. Electrophysiological studies can be used to identify the presence of a pathophysiological substrate that establishes risk for developing symptomatic arrhythmias. Its place in the evaluation of unexplained syncope in the individual patient depends on the relative probabilities of a cardiac cause, as determined by clinical judgment.

During electrophysiological studies, attempts are made to evaluate sinus node function, AV conduction, and inducibility of supraventricular and ventricular tachyarrhythmias. In patients with structural heart disease, the most common abnormality identified during electrophysiological testing is VT. Less frequently, His-Purkinje block and sinus node dysfunction are encountered. Induction of sustained monomorphic VT, paroxysmal supraventricular tachycardia, His-Purkinje block, and evidence of sinus node dysfunction may have diagnostic and prognostic value in patients with unexplained syncope. Induction of atrial fibrillation, atrial flutter, nonsustained VT, polymorphic VT, and ventricular fibrillation induced by aggressive protocols may be nonspecific and must be interpreted carefully.

In general, among patients without structural heart disease who have a normal ECG, the diagnostic yield of electrophysiological studies is low,[81] and head-up tilt testing may provide useful diagnostic information.[74] In contrast, in patients with underlying structural heart disease, such as prior MI, particularly if the signal-averaged ECG shows late potentials, an arrhythmia is more likely to be the cause of syncope, and electrophysiological testing has a high priority. However, the possibility that the patient with structural heart disease may also have neurocardiogenic syncope should not be overlooked.

Recommendations for Electrophysiological Studies

Class I

Patients with suspected structural heart disease and syncope that remains unexplained after appropriate evaluation

Class II

Patients with recurrent unexplained syncope without structural heart disease and a negative head-up tilt test

Class III

Patients with a known cause of syncope for whom treatment will not be guided by electrophysiological testing

XI. Role of Electrophysiological Study in Survivors of Cardiac Arrest

Patients resuscitated from cardiac arrest not associated with a new Q-wave MI remain at high risk for recurrent cardiac arrest and sudden cardiac death during long-term follow-up.[86-90] Although reported to be 30% at 1 year of follow-up and 45% at 2 years of follow-up in the 1970s,[88,89] the current magnitude of risk is unknown. Risk of recurrent cardiac arrest may be declining due to the combination of the overall decrease in cardiovascular mortality, more aggressive therapy targeted to the pathophysiology and manifestations of the underlying heart diseases (eg, thrombolytic and coronary artery revascularization strategies), and therapy aimed specifically at cardiac arrhythmias. Current natural history figures are unavailable because of the failure to control for these variables, but these patients are still considered to be at excess risk for recurrent cardiac arrest.[91]

In the absence of antiarrhythmic drug therapy, ventricular tachyarrhythmias can be initiated during electrophysiological studies in 70% to 80% of patients resuscitated from cardiac arrest. Sustained monomorphic VT is inducible in 36% to 51% of the patients, with various proportions of ventricular fibrillation, monomorphic or polymorphic VT degenerating to fibrillation, and nonsustained VT distributed among the remainder.[92-97] Among the subgroup of cardiac arrest survivors in whom sustained monomorphic VT is identified as the electrical mechanism initiating cardiac arrest, the percentage of patients with inducible monomorphic VT is considerably higher. The ability to prevent induction of a previously inducible sustained ventricular tachyarrhythmia, as a result of pharmacological or surgical intervention, is associated with a more favorable outcome during follow-up than is failure to identify a successful therapeutic end point.[96-102] Stratification by ejection fraction is a major modifier of outcome after successful suppression of inducibility.[97]

Identification of a drug that suppresses inducible VT or fibrillation has been reported in 26% to 80% of survivors of cardiac arrest,[92-97] with most studies clustering toward the lower rather than higher percentages. Survivors of cardiac arrest whose arrhythmias remain inducible at the time of discharge from the hospital are at more than twice the risk of recurrent cardiac arrest compared with those with ventricular tachyarrhythmias that are rendered noninducible.[97]

The value of preoperative and postoperative studies in cardiac arrest survivors who undergo surgical therapy depends on the nature of the arrhythmias. Among patients with ventricular fibrillation associated with a transient ischemic mechanism, electrophysiological studies are of limited usefulness; however, in patients who have inducible monomorphic VT before surgery and who undergo map-guided antiarrhythmic surgical procedures, postoperative studies are useful for predicting freedom from ventricular tachyarrhythmias during follow-up.[103,104]

The significance of failure to induce ventricular tachyarrhythmias during baseline electrophysiological studies in the absence of antiarrhythmic drugs in survivors of cardiac arrest has been a source of debate in the past. However, it is now generally accepted that patients with depressed left ventricular function and no obvious reversible cause of arrhythmias (eg, ischemia) remain at risk for recurrent cardiac arrest, despite failure to induce ventricular tachyarrhythmias at baseline.[97] In contrast, patients with a documented ischemic mechanism for cardiac arrest who have a normal or near-normal ejection fraction and do not have inducible ventricular tachyarrhythmias during electrophysiological testing remain at low risk after treatment of the underlying ischemia. Among patients who are candidates for implantable cardioverter-defibrillator devices, preimplant studies are useful for determining the most appropriate type of device for implantation. The nature of induced arrhythmias, in terms of electrical and hemodynamic stability and ability to terminate the arrhythmia by pacing, will guide the choice of device therapy.[105]

Among patients who have unexplained cardiac arrest in the absence of structural heart disease, electrophysiological studies are used to identify a therapeutic target, but the yield of such studies is considerably lower than in those with structural heart disease.[106]

Recommendations for Electrophysiological Studies

Class I

(1) Patients surviving cardiac arrest without evidence of an acute Q-wave MI

(2) Patients surviving cardiac arrest occurring more than 48 hours after the acute phase of MI in the absence of a recurrent ischemic event

Class II

(1) Patients surviving cardiac arrest caused by bradyarrhythmia

(2) Patients surviving cardiac arrest thought to be associated with a congenital repolarization abnormality (long QT syndrome) in whom the results of noninvasive diagnostic testing are equivocal

Class III

(1) Patients surviving a cardiac arrest that occurred during the acute phase (<48 hours) of MI

(2) Patients with cardiac arrest resulting from clearly definable specific causes such as reversible ischemia, severe valvular aortic stenosis, or noninvasively defined congenital or acquired long QT syndrome

XII. Role of Electrophysiological Study in Patients With Unexplained Palpitations

Long-term ambulatory recording is the most useful procedure for documenting cardiac rhythm associated with palpitations. The recording can be a continuous 24-hour recording if palpitations occur daily, or a loop or event recording if infrequent.[107] Electrophysiological studies are used if recording attempts fail to provide an answer. The sensitivity of electrophysiological studies is low in patients with unexplained palpitations.

Recommendations for Electrophysiological Studies

Class I

(1) Patients with palpitations who have a pulse rate documented by medical personnel as inappropriately rapid

JACC Vol. 26, No. 2
August 1995:555–73

and in whom ECG recordings fail to document the cause of the palpitations

(2) Patients with palpitations preceding a syncopal episode

Class II

Patients with clinically significant palpitations, suspected to be of cardiac origin, in whom symptoms are sporadic and cannot be documented. Studies are performed to determine the mechanisms of arrhythmias, direct or provide therapy, or assess prognosis.

Class III

Patients with palpitations documented to be due to extracardiac causes (eg, hyperthyroidism)

XIII. Role of Electrophysiological Study in Guiding Drug Therapy

Electrophysiological studies allow serial assessment of drug-induced changes in conduction and refractoriness in cardiac tissues and in properties of arrhythmias, including inducibility, and, if inducible, rate, morphology, and hemodynamic consequences. After a baseline study (preferably when the patient is off medication) during which an arrhythmia is induced, a drug is administered, and electrical stimulation is repeated. It has been proposed that drug-induced suppression of ability to reinduce the arrhythmia will predict freedom from recurrent arrhythmias. Conversely, if the arrhythmia remains inducible, the probability of arrhythmia recurrence is higher than if suppression had been achieved. This approach has been used primarily in patients with sustained VT and in survivors of cardiac arrest, but similar studies in patients with supraventricular arrhythmias are also possible.

Ventricular Arrhythmias

Sustained VT can be initiated with programmed stimulation in over 90% of patients with prior MI and a history of sustained monomorphic VT. Induction rates are lower in patients whose clinical presentation was cardiac arrest, in patients with nonsustained VTs, and with other forms of heart disease. If a sustained arrhythmia can be initiated at baseline, observational studies have indicated that arrhythmia-free survival is higher among patients in whom drug-induced suppression of arrhythmia induction was achieved at a follow-up study.[96-102] It is not clear whether the higher arrhythmia-free survival rate is due to the effects of the antiarrhythmic drug or whether this response to electrophysiological testing merely selects out patients at lower risk. Among patients in whom VT remains inducible, characteristics of the arrhythmia induced during electrophysiological study predict features of future recurrences.[101,102] When the tachycardia is not significantly modified by drug effect, an adverse risk for both recurrent tachycardias and mortality is predicted. However, when the tachycardia cycle length is prolonged by 100 milliseconds or more and the induced tachycardia is hemodynamically stable, mortality outcome is similar to risk predicted by successful drug therapy, while the risk of recurrent tachycardia parallels the data for drug failure.[101]

Alternative approaches for guidance of antiarrhythmic drug therapy are available. In selected patients, empiric therapy with a β-adrenergic blocker[108] or amiodarone[109] may be useful and, in some patients, superior to therapy with other agents guided by serial electrophysiological study. Sotalol has also been shown to be superior to conventional antiarrhythmic agents in one study.[110] Two randomized trials[111,112] and several observational studies[113-116] have compared serial ambulatory monitoring and serial electrophysiological testing as methods for selecting antiarrhythmic drug therapy. Data from these trials are discordant because of methodological limitations of the studies, the significant long-term toxicity of the drugs tested, and the relative inefficacy of most drugs when assessed by serial electrophysiological testing. Hence, superiority of either technique is still unclear, and at present both invasive and noninvasive methods can be considered part of the evaluation for therapy.

Supraventricular Arrhythmias

The effects of antiarrhythmic drugs on tissues involved in supraventricular arrhythmias can be assessed with electrophysiological studies. Factors associated with clinical success in patients with paroxysmal supraventricular tachycardia due to atrioventricular nodal reentry or atrioventricular reentry include induction of block or marked lengthening of the refractory period in one limb of a reentrant circuit, suppression of the ability to initiate sustained arrhythmia, and reduction of maximum ventricular rates during atrial fibrillation in patients with preexcitation.[117-119] Isoproterenol has reversed many of these antiarrhythmic drug effects, thus limiting the predictive value of these acute observations.[120] Only limited data concerning the predictive value of suppression of induction of other atrial arrhythmias are available.

Recommendations for Electrophysiological Studies

Class I

(1) Patients with sustained VT or cardiac arrest, especially those with prior MI

(2) Patients with AVNRT, AV reentrant tachycardia using an accessory pathway, or atrial fibrillation associated with an accessory pathway, for whom chronic drug therapy is planned

Class II

(1) Patients with sinus node reentrant tachycardia, atrial tachycardia, atrial fibrillation, or atrial flutter without ventricular preexcitation syndrome, for whom chronic drug therapy is planned

(2) Patients with arrhythmias not inducible during control electrophysiological study for whom drug therapy is planned

Class III

(1) Patients with isolated atrial or ventricular premature complexes

(2) Patients with ventricular fibrillation with a clearly identified reversible cause

XIV. Role of Electrophysiological Study in Patients Who Are Candidates for or Who Have Implantable Electrical Devices

The role of electrophysiological studies in determining the need for permanent pacing has already been discussed in the sections on sinus node dysfunction (section II) and AV block (sections III and IV). Electrophysiological studies can also be used before pacemaker implantation to provide physiological data that can influence the mode, site(s), and programmable pacing functions to be chosen in the long-term pacing prescription.[121-123] Most modern permanent pacemakers have sophisticated telemetry capabilities that allow noninvasive assessment of many aspects of pacemaker function after insertion. Additional invasive electrophysiological procedures are only needed when different sites of stimulation are required or the implanted system cannot replicate the modality of pacing to be tested.

Implantable electrical devices are an important therapeutic option in many patients with tachyarrhythmias. Some arrhythmias (eg, torsade de pointes and atrial fibrillation in patients with sinus node dysfunction) can arise in the setting of bradycardias, and standard bradycardia pacing may be helpful in preventing future episodes.[124,125] In selected individuals, dual-chamber pacemakers programmed with short AV delays have been used to prevent some AV reentrant tachycardias.[126] Antitachycardia pacing with extrastimuli or bursts can be used to terminate many supraventricular and ventricular arrhythmias. However, because antitachycardia pacing can accelerate the original tachycardia, automatic antitachycardia pacing for ventricular arrhythmias is not advisable unless automatic defibrillation back-up is available. In patients with supraventricular arrhythmias, risks associated with the potential induction of atrial fibrillation must be considered before antitachycardia pacing is prescribed. Electrophysiological studies performed before device implantation may be used to assess the potential efficacy and risks associated with antitachycardia pacing.

Implantable cardioverter-defibrillators (ICDs) have been in use for more than 15 years.[127] There is general consensus that they prevent sudden arrhythmic deaths, but their effects on total mortality, particularly in patients with depressed ventricular function, are still uncertain.[128,129] Advances in ICD technology have been rapid, and current devices often include antitachycardia pacing, bradycardia pacing, low energy cardioversion, high energy defibrillation, sophisticated diagnostic functions, capability to perform noninvasive programmed stimulation, and transvenous or subcutaneous lead systems.[130,131]

Data obtained during electrophysiological studies are used to guide selection of the appropriate implantable electric device and for programming long-term device settings. Electrophysiological studies are appropriate before implantation to assess the characteristics of the arrhythmia or arrhythmias to be treated, at implantation to assess the efficacy of the device, and after implantation to confirm continued effectiveness, particularly if changes in the patient's status or therapy that might affect the function of the device have occurred.

Recommendations for Electrophysiological Study

Class I

(1) Patients with tachyarrhythmias, before and during implantation, and final (predischarge) programming of an electrical device to confirm its ability to perform as anticipated

(2) Patients with an implanted electrical antitachyarrhythmia device in whom changes in status or therapy may have influenced the continued safety and efficacy of the device

(3) Patients who have a pacemaker to treat a bradyarrhythmia, and receive a cardioverter-defibrillator, to test for device interactions

Class II

Patients with previously documented indications for pacemaker implantation to test for the most appropriate long-term pacing mode and sites to optimize symptomatic improvement and hemodynamics

Class III

Patients who are not candidates for device therapy

XV. Indications for Catheter Ablation Procedures

Catheter ablation was introduced in the early 1980s and has become the treatment of choice for some arrhythmias and an important consideration for others. More than 10 000 ablation procedures were performed in the United States in 1992, with complication rates as low as 2% in some patient groups.[132] Catheter ablation has largely supplanted open-heart surgical procedures for several types of arrhythmias and is an acceptable alternative to long-term drug therapy. The role of catheter ablation as primary therapy for several arrhythmias has been described in position papers or technology assessments by the American Medical Association,[133] the American College of Cardiology,[134] and the North American Society of Pacing and Electrophysiology.[135] The use of direct current shocks for ablation has largely been relegated to a secondary role. Other energy sources are being evaluated, but these guidelines relate primarily to the use of radiofrequency current for ablation.

Radiofrequency Catheter Ablation or Modification of Atrioventicular Junction for Ventricular Rate Control of Atrial Tachyarrhythmias

Catheter ablation of the AV junction (producing complete AV block) is well established as a means of controlling ventricular response in patients with poor rate control who are receiving medical therapy. Recently, selective ablation in the region of the posteroseptal and midseptal approaches to the AV node has been used to control ventricular response without producing complete AV block.[136]

The efficacy of producing complete AV block by radiofrequency ablation of the AV junction varies from 70% to 95% and is usually 90% or more.[132,137-141] Complication rates are generally less than 2%, and procedure-related deaths are estimated at 0.1%.[132] Late sudden death may follow AV junction ablation but may be less with radiofrequency ablation than DC shock ablation.[142] Many patients undergoing ablation of the AV junction have severely compromised cardiac function, and it is uncertain whether

the late deaths are directly related to the ablative procedure or the underlying myocardial disease.

Recommendations for Radiofrequency Catheter Ablation and Modification of Atrioventricular Junction

Class I

(1) Patients with symptomatic atrial tachyarrhythmias who have inadequately controlled ventricular rates *unless* primary ablation of the atrial tachyarrhythmia is possible

(2) Patients with symptomatic atrial tachyarrhythmias such as those above but when drugs are not tolerated or the patient does not wish to take them, even though the ventricular rate can be controlled

(3) Patients with symptomatic nonparoxysmal junctional tachycardia that is drug resistant, drugs are not tolerated, or the patient does not wish to take them

(4) Patients resuscitated from sudden cardiac death due to atrial flutter or atrial fibrillation with a rapid ventricular response in the absence of an accessory pathway

Class II

Patients with a dual-chamber pacemaker and pacemaker-mediated tachycardia that cannot be treated effectively by drugs or by reprogramming the pacemaker

Class III

Patients with atrial tachyarrhythmias responsive to drug therapy acceptable to the patient

Radiofrequency Catheter Ablation for Atrioventricular Nodal Reentrant Tachycardia

The AV node includes "fast" pathways with atrial connections located anteriorly and "slow" pathways with atrial connections located posteriorly. In the most common type of AVNRT, the slow pathway is used for anterograde conduction and the fast pathway is used for retrograde conduction. Both pathways are needed to maintain AVNRT. The atrial connection of either the fast or slow pathway can be ablated, thereby eliminating AVNRT. Slow pathway ablation is preferred because of a lower incidence of producing AV block, a greater likelihood of maintaining a normal PR interval during sinus rhythm, and its efficacy in the atypical forms of AVNRT. The 1992 NASPE survey[132] included 3052 patients undergoing slow pathway ablation with a success rate of 96% and 255 patients undergoing fast pathway ablation with a success rate of 90%. Recurrence of AVNRT after an initially successful procedure has an estimated frequency of about 5%. Overall complications were 0.96%; no procedure-related deaths were reported.

Recommendations for Radiofrequency Catheter Ablation for Atrioventricular Nodal Reentrant Tachycardia

Class I

Patients with symptomatic sustained AVNRT that is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy

Class II

(1) Patients with sustained AVNRT identified during electrophysiological study or catheter ablation of another arrhythmia

(2) The finding of dual AV nodal pathway physiology and atrial echoes but without AVNRT during electrophysiological study in patients suspected of having AVNRT clinically

Class III

(1) Patients with AVNRT responsive to drug therapy that is well tolerated and preferred by the patient to ablation

(2) The finding of dual AV nodal pathway physiology (with or without echo complexes) during electrophysiological study in patients in whom AVNRT is not suspected clinically

Radiofrequency Catheter Ablation of Atrial Tachycardia, Flutter, and Fibrillation

Atrial tachycardia and atrial flutter were reported together in the NASPE survey[132]; the success rate was 75% in 371 patients with a complication rate of 0.81% and no reported deaths. There are increasing numbers of publications related to ablation of atrial tachycardias,[132,137,143-150] including tachycardia in the region of the sinus node and ablation of inappropriate sinus tachycardia.[147] Radiofrequency ablation has also been effective in eliminating common atrial flutter.[143,149] Although surgical procedures involving incision and isolation of atrial myocardium have been devised to eliminate atrial fibrillation and their feasibility has been demonstrated, catheter ablation techniques for eliminating atrial fibrillation are at a relatively early stage of development, but preliminary success has been reported.[151,152]

Recomendations for Radiofrequency Catheter Ablation of Atrial Tachycardia, Flutter, and Fibrillation

Class I

(1) Patients with atrial tachycardia that is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy

(2) Patients with atrial flutter that is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy[153]

Class II

(1) Atrial flutter/atrial tachycardia associated with paroxysmal atrial fibrillation when the tachycardia is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy

(2) Patients with atrial fibrillation and evidence of a localized site(s) of origin when the tachycardia is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy

Class III

(1) Patients with atrial arrhythmia that is responsive to drug therapy, well tolerated, and preferred by the patient to ablation

(2) Patients with multiform atrial tachycardia

Radiofrequency Catheter Ablation of Accessory Pathways

The safety, efficacy[132,137,154-160] and cost-effectiveness[161] of radiofrequency ablation of an accessory AV pathway has

made ablation the treatment of choice in most patients who have AV reentrant tachycardia or atrial fibrillation (or other atrial tachyarrhythmias) associated with a rapid ventricular response via the accessory pathway. Results from the literature[133,135,137,154-161] and the NASPE survey[132] are comparable. The NASPE survey reports success rates of 91% in 2527 left free-wall accessory pathways, 87% in 1279 septal pathways, and 82% in 715 right free-wall pathways; overall rates of complications and death were 2.1% and 0.2%, respectively. Complications include the possibility of valve damage, pericardial tamponade, AV block, and pulmonary or systemic emboli. Rare late deaths have been reported.[137]

Recommendations for Radiofrequency Catheter Ablation of Accessory Pathways

Class I

(1) Patients with symptomatic AV reentrant tachycardia that is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy

(2) Patients with atrial fibrillation (or other atrial tachyarrhythmia) and a rapid ventricular response via the accessory pathway when the tachycardia is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy

Class II

(1) Patients with AV reentrant tachycardia or atrial fibrillation with rapid ventricular rates identified during electrophysiological study of another arrhythmia

(2) Asymptomatic patients with ventricular preexcitation whose livelihood or profession, important activities, insurability, or mental well being or the public safety would be affected by spontaneous tachyarrhythmias or the presence of the ECG abnormality

(3) Patients with atrial fibrillation and a controlled ventricular response via the accessory pathway

(4) Patients with a family history of sudden cardiac death

Class III

Patients who have accessory pathway–related arrhythmias that are responsive to drug therapy, well tolerated, and preferred by the patient to ablation

Radiofrequency Catheter Ablation of Ventricular Tachycardia

Radiofrequency ablation of VT has been used with varying degrees of success in patients with ischemic disease,[132,162-165] cardiomyopathy,[132] bundle branch reentry,[166,167] and various forms of idiopathic VT.[50,168-170] In the NASPE survey,[132] there was a successful ablation rate of 71% overall in 429 patients with VT ; 85% in 224 patients with structurally normal hearts; 54% in 115 with ischemic heart disease; and 61% in 90 with idiopathic cardiomyopathy. Complications occurred in 3%, with no reported deaths. Mapping and ablation techniques differ, depending on the type of VT. In patients without structural heart disease, only a single VT is usually present, and catheter ablation is curative. In patients with extensive structural heart disease, especially those with prior MI, multiple VTs are often present. Catheter ablation of a single VT in such patients may be only palliative and may not eliminate the need for other antiarrhythmic therapy.

Recommendations for Radiofrequency Catheter Ablation of Ventricular Tachycardia

Class I

(1) Patients with symptomatic sustained monomorphic VT when the tachycardia is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy

(2) Patients with bundle branch reentrant ventricular tachycardia

(3) Patients with sustained monomorphic VT and an ICD who are receiving multiple shocks not manageable by reprogramming or concomitant drug therapy

Class II

Nonsustained VT that is symptomatic when the tachycardia is drug resistant or the patient is drug intolerant or does not desire long-term drug therapy

Class III

(1) Patients with VT that is responsive to drug, ICD, or surgical therapy and that therapy is well tolerated and preferred by the patient to ablation

(2) Unstable, rapid, multiple, or polymorphic VT that cannot be adequately localized by current mapping techniques

(3) Asymptomatic and clinically benign nonsustained VT

XVI. Role of Electrophysiological Study in Pediatric Patients: Differences From Adults

Although there are nuances specific to pediatric patients, performance and interpretation of intracardiac electrophysiological studies in children are generally similar to those in adults. Indications for electrophysiological studies in children are also similar to general indications in adults. However, there are differences in some areas. The patient's age can influence indications for an electrophysiological study and dictate technical decisions, as can the presence of associated congenital heart lesions.

Need for Sedation

Small children, and even adolescents, have special requirements for sedation. The electrophysiological effects of sedation can be vagolytic (meperidine and promethazine) or sympathomimetic (ketamine).[171,172] In a child, physiological conditions may change throughout a study with the addition of different types of sedation as well as different states of wakefulness. For this reason, tests of sinus node function and AV conduction as well as refractory periods of accessory connections may be less reproducible and potentially less valid in children than in adults.

Prognostic Testing in "High-Risk" Groups

Some children, such as those who have had repair of some types of congenital heart disease, are thought to be at high risk for arrhythmic death.[173-175] No randomized studies have been carried out to determine if intervention alters

outcome in such patients. Some pediatric cardiologists have advocated the use of electrophysiological studies to identify postsurgical patients who may be at higher risk for sudden death.[176] Although most ventricular ectopy in children with normal hearts is benign, nonsustained VT or premature ventricular complexes that are not suppressed with exercise may be the first sign of an otherwise subclinical myopathy or myocarditis. Some authors have advocated electrophysiological studies in these patients.[177] The risk of some arrhythmias may be greater in children than in adults because the adult population represents survivors. The incidence of sudden death in children is low, so prospective data on this subject are limited.[178]

Pediatric Tachycardias

Incessant supraventricular tachycardias can lead to cardiomyopathy that occasionally is severe enough to require cardiac transplantation.[179] The major causes are atrial automatic tachycardia and the permanent form of junctional reciprocating tachycardia and atypical AV node reentry. These conditions are relatively uncommon in adults but more common in children, and atrial automatic tachycardia can be confused with sinus tachycardia. In a child with dilated cardiomyopathy believed to have "sinus tachycardia," it may be important to perform electrophysiological mapping to distinguish chronic atrial tachycardia from sinus tachycardia. Electrophysiological studies and mapping followed by ablation have resulted in return of normal cardiac function.[180] Electrophysiological studies with radiofrequency catheter ablation have been very successful in treating each mechanism of supraventricular tachycardia in infants and children, with the exception of atrial fibrillation.[181] The greatest number of ablations have been performed for reentry using an accessory connection and AV nodal reentry.[182] Certain VTs and atrial flutter in children may also be treated by ablation. While indications for ablation are generally similar in pediatric patients and adults, recent data from animals suggest that the ablation lesion can enlarge in children as they grow. Therefore, until longer follow-up data are available, the long-term risk of ablation, particularly in younger children, is not well established.

Complete Atrioventricular Block

Congenital complete AV block most often occurs with a narrow QRS escape rhythm. Electrophysiological studies have not been demonstrated clinically useful in this situation. However, if congenital complete AV block occurs with a wide QRS escape rhythm, such studies could provide data to determine the site of block and the presence of infranodal disease. Acquired complete AV block in children is considered an indication for a permanent pacemaker, and electrophysiological studies are not necessary. Electrophysiological studies have not been beneficial in predicting prognosis in asymptomatic patients with surgically acquired bifascicular block. They may be useful in some postoperative patients with transient complete AV block.

Recommendations for Electrophysiological Studies

Class I

(1) Pediatric patients with conditions or characteristics similar to those described in the sections on adults

(2) Patients with an undiagnosed narrow QRS tachycardia that cannot be distinguished from sinus tachycardia

Class II

(1) Pediatric patients with conditions or characteristics similar to those described in the sections on adults

(2) Asymptomatic patients possibly at high risk for sudden arrhythmic death, such as the postoperative patient with complex congenital heart disease or a normal heart with complex ventricular arrhythmias (nonsustained VT or premature ventricular complexes that fail to suppress during exercise)

(3) Patients with congenital complete AV block and wide QRS escape rhythm

Class III

(1) Pediatric patients with conditions or characteristics similar to those described in the sections on adults

(2) Patients with congenital complete AV block and narrow QRS escape rhythm

(3) Patients with acquired complete AV block

(4) Asymptomatic patients with surgically induced bifascicular block

Appendix

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